STN SEARCH 10/735,973 9/30/2006
=> Index bioscience medicine

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 15:54:39 ON 30 SEP 2006

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409 FILE NLDB

68 FILES HAVE ONE OR MORE ANSWERS, 71 FILES SEARCHED IN STNINDEX

L1 QUE (PHOSPHODIESTERASE OR PDE4 OR PDE4D3)

=> d rank

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F12	7277	GENBANK
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F14	5403	ESBIOBASE
F15	4422	LIFESCI
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F18	3115	WPINDEX
F19	2268	IFIPAT
F20	1758	DDFB
F21	1758	DRUGB
F22	1686	JICST-EPLUS
F23	1366	CABA
F24	1213	USPAT2
F25	1069	PROMT

=> file F1-f8, f10, f11, f14, f17

FILE 'CAPLUS' ENTERED AT 15:56:51 ON 30 SEP 2006
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FILE 'WPIDS' ENTERED AT 15:56:51 ON 30 SEP 2006
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=> S L1
L2 175441 L1

=> S (isozyme or isoenzyme) (s) L2
L3 3250 (ISOZYME OR ISOENZYME) (S) L2

=> S (modif? or mutat? or alter? or mutant or variant) (s) L3
10 FILES SEARCHED...
L4 177 (MODIF? OR MUTAT? OR ALTER? OR MUTANT OR VARIANT) (S) L3

=> S (Aggregat? or solub? or insolub?)(s)L4
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PROCESSING COMPLETED FOR L5
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=> d ibib abs L6 1-12

L6 ANSWER 1 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2006:53958 USPATFULL <<LOGINID::20060930>>
TITLE: Differential expression of molecules associated with acute stroke
INVENTOR(S): Baird, Alison E., Bethesda, MD, UNITED STATES
Moore, David F., Rockville, MD, UNITED STATES
Goldin, Ehud, Rockville, MD, UNITED STATES
PATENT ASSIGNEE(S): The Gov. of the U.S.A as represented by the Secretary of the Dept. of Health & Human Services (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006046259	A1	20060302
APPLICATION INFO.:	US 2005-155835	A1	20050617 (11)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2005-US18744, filed on 27 May 2005, PENDING

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-575279P	20040527 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: KLARQUIST SPARKMAN, LLP, 121 S.W. SALMON STREET, SUITE #1600, ONE WORLD TRADE CENTER, PORTLAND, OR, 97204-2988, US
NUMBER OF CLAIMS: 75
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 1 Drawing Page(s)
LINE COUNT: 6359
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods are provided for evaluating a stroke, for example for determining whether a subject has had an ischemic stroke, determining the severity or likely neurological recovery of a subject who has had an ischemic stroke, and determining a treatment regimen for a subject who

has had an ischemic stroke, as are arrays and kits that can be used to practice the methods. In particular examples, the method includes screening for expression in ischemic stroke related genes (or proteins), such as white blood cell activation and differentiation genes (or proteins), genes (or proteins) related to hypoxia, genes (or proteins) involved in vascular repair, and genes (or proteins) related to a specific peripheral blood mononuclear cell (PBMC) response to the altered cerebral microenvironment. Also provided are methods of identifying one or more agents that alter the activity (such as the expression) of an ischemic stroke-related molecule.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 2 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2006:9955 USPATFULL <<LOGINID::20060930>>
TITLE: Identification of tissue/cell specific marker genes and use thereof
INVENTOR(S): Brunner, Andreas, Oberembrach, SWITZERLAND
Hagg, Rupert, Bassesdorf, SWITZERLAND
Tommasini, Roberto, Uster, SWITZERLAND

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2006008803	A1	20060112
APPLICATION INFO.:	US 2003-517756	A1	20030612 (10)
	WO 2003-CH379		20030612
			20050802 PCT 371 date

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-388994P	20020614 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: LADAS & PARRY, 26 WEST 61ST STREET, NEW YORK, NY, 10023, US
NUMBER OF CLAIMS: 38
EXEMPLARY CLAIM: 1-29
NUMBER OF DRAWINGS: 6 Drawing Page(s)
LINE COUNT: 4438
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A cartilage array comprises a plurality of different polynucleotide probe spots stably associated with a solid surface of a carrier, whereby each of said spots is made of a unique polynucleotide that corresponds to one specific cartilage marker gene. Said specific cartilage marker genes preferably are at least in part selected from a group of 467 genes that could be shown to be cartilage related.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 3 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2005:298974 USPATFULL <<LOGINID::20060930>>
TITLE: Method for diagnosing pancreatic cancer
INVENTOR(S): Nakamura, Yusuke, Yokohama-shi, JAPAN
Katagiri, Toyomasa, Shinagawa-ku, JAPAN
Nakagawa, Hidewaki, Shinagawa-ku, JAPAN
PATENT ASSIGNEE(S): Oncotherapy Science, Inc., Kawasaki-shi, JAPAN (non-U.S. corporation)
The University of Tokyo, Bunkyo-ku, JAPAN (non-U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005260639	A1	20051124
APPLICATION INFO.:	US 2005-90739	A1	20050324 (11)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. WO 2003-JP11817, filed on 17 Sep 2003, UNKNOWN

	NUMBER	DATE
PRIORITY INFORMATION:	US 2004-555809P	20040324 (60)

US 2003-450889P 20030228 (60)
US 2002-414872P 20020930 (60)
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: TOWNSEND AND TOWNSEND AND CREW, LLP, TWO EMBARCADERO CENTER, EIGHTH FLOOR, SAN FRANCISCO, CA, 94111-3834, US
NUMBER OF CLAIMS: 60
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 16 Drawing Page(s)
LINE COUNT: 6547
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Objective methods for detecting and diagnosing pancreatic cancer (PNC) are described herein. In one embodiment, the diagnostic method involves determining the expression level of PNC-associated gene that discriminates between PNC cells and normal cells. The present invention further provides methods of screening for therapeutic agents useful in the treatment of pancreatic cancer, methods of treating pancreatic cancer and method of vaccinating a subject against pancreatic cancer.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 4 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2005:104584 USPATFULL <<LOGINID::20060930>>
TITLE: Treatment of respiratory diseases with anti-IL-2 receptor antibodies
INVENTOR(S): Shames, Richard S., Palo Alto, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005089517	A1	20050428
APPLICATION INFO.:	US 2004-947432	A1	20040921 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-505883P	20030923 (60)
	US 2004-552974P	20040312 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: HOWREY SIMON ARNOLD & WHITE, LLP, c/o IP DOCKETING DEPARTMENT, 2941 FAIRVIEW PARK DRIVE, SUITE 200, FALLS CHURCH, VA, 22042-2924, US
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 4 Drawing Page(s)
LINE COUNT: 2181
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention provides a method of treating respiratory and allergic diseases. In particular, it provides a method for the treatment of asthma comprising administering to a subject a therapeutically effective amount of a pharmaceutical formulation comprising an antibody, wherein said antibody binds to IL-2 receptor.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 5 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2005:69014 USPATFULL <<LOGINID::20060930>>
TITLE: Electromagnetic activation of gene expression and cell growth
INVENTOR(S): George, Frank R., Scottsdale, AZ, UNITED STATES
Moffett, John, Phoenix, AZ, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2005059153	A1	20050317
APPLICATION INFO.:	US 2004-759526	A1	20040116 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2003-509061P	20030122 (60)

DOCUMENT TYPE: Utility

FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Cathryn Campbell, McDERMOTT, WILL & EMERY, Suite 700, 4370 La Jolla Village Drive, San Diego, CA, 92122
NUMBER OF CLAIMS: 62
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 29 Drawing Page(s)
LINE COUNT: 2183
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention is directed to a method for accelerating the cell cycle by delivering to a cell an effective amount of electromagnetic energy. The invention also provides a method for activating a cell cycle regulator by delivering to a cell an effective amount of electromagnetic energy. Also provided by the invention is a method for activating a signal transduction protein; a method for activating a transcription factor; a method for activating a DNA synthesis protein; and a method for activating a Receptor. A method for inhibiting an angiotensin receptor as well as a method for reducing inflammation also are provided by the present invention. The invention also is directed to a method for replacing damaged neuronal tissue as well as a method for stimulating growth of administered cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 6 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2004:133338 USPATFULL <<LOGINID::20060930>>
TITLE: Targets for therapeutic intervention identified in the mitochondrial proteome
INVENTOR(S): Ghosh, Soumitra S., San Diego, CA, UNITED STATES
Fahy, Eoin D., San Diego, CA, UNITED STATES
Zhang, Bing, Spring, TX, UNITED STATES
Gibson, Bradford W., Berkeley, CA, UNITED STATES
Taylor, Steven W., San Diego, CA, UNITED STATES
Glenn, Gary M., Encinitas, CA, UNITED STATES
Warnock, Dale E., San Diego, CA, UNITED STATES
Gaucher, Sara P., Castro Valley, CA, UNITED STATES
PATENT ASSIGNEE(S): MitoKor Inc., San Diego, CA, UNITED STATES, 92121 (U.S. corporation)
The Buck Institute for Age Research, Novato, CA, UNITED STATES, 94948-0638 (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2004101874	A1	20040527
APPLICATION INFO.:	US 2003-408765	A1	20030404 (10)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2002-412418P	20020920 (60)
	US 2002-389987P	20020617 (60)
	US 2002-372843P	20020412 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: SEED INTELLECTUAL PROPERTY LAW GROUP PLLC, 701 FIFTH AVE, SUITE 6300, SEATTLE, WA, 98104-7092
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 5 Drawing Page(s)
LINE COUNT: 5998
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Mitochondrial targets for drug screening assays and for therapeutic intervention in the treatment of diseases associated with altered mitochondrial function are provided. Complete amino acid sequences [SEQ ID NOS:1-3025] of polypeptides that comprise the human heart mitochondrial proteome are provided, using fractionated proteins derived from highly purified mitochondrial preparations, to identify previously unrecognized mitochondrial molecular components.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 7 OF 12 USPATFULL on STN

ACCESSION NUMBER: 2004:141124 USPATFULL <<LOGINID::20060930>>
TITLE: Diagnostics and therapeutics for an obstructive airway disease
INVENTOR(S): Duff, Gordon W., Sheffield, UNITED KINGDOM
di Giovine, Francesco S., Ranmoor, UNITED KINGDOM
Barnes, Peter J., London, UNITED KINGDOM
Lim, Samson, Concord, AUSTRALIA
PATENT ASSIGNEE(S): Interleukin Genetics, Inc., Waltham, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6746839	B1	20040608
APPLICATION INFO.:	US 2000-584950		20000601 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1998-5923, filed on 12 Jan 1998, now patented, Pat. No. US 6140047, issued on 31 Oct 2000
DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Fredman, Jeffrey
ASSISTANT EXAMINER: Chakrabarti, Arun Kr.
LEGAL REPRESENTATIVE: Mintz Levin, Elrifi, Ivor R., Kozakiewicz, Cynthia A.
NUMBER OF CLAIMS: 49
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 15 Drawing Figure(s); 15 Drawing Page(s)
LINE COUNT: 3470
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods and kits for detecting polymorphisms that are predictive of a subject's susceptibility to developing an obstructive airway disease, such as asthma, as well as for determining the relative severity of the disease are described. Assays for identify therapeutics are also described.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 8 OF 12 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2004-122028 [12] WPIDS
CROSS REFERENCE: 2000-475722 [41]; 2000-491087 [43]; 2000-491116 [43]; 2000-491166 [43]; 2000-572155 [53]; 2001-016296 [02]; 2002-282788 [33]; 2002-507086 [54]; 2002-682228 [73]; 2002-723387 [78]; 2003-030038 [02]; 2003-229203 [22]
DOC. NO. CPI: C2004-048798
TITLE: Identifying breast cancer or breast precancer in humans comprises providing a ductal fluid sample from one duct of a breast of a patient, and examining the ductal fluid sample for the presence of a marker (e.g. a DNA or a protein).
DERWENT CLASS: B04 D16
INVENTOR(S): HUNG, D T
PATENT ASSIGNEE(S): (CYTY-N) CYTYC HEALTH CORP
COUNTRY COUNT: 1
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2004018546	A1	20040129 (200412)*			18

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2004018546	A1 Provisional	US 1999-117281P	19990126
	CIP of	US 1999-313463	19990517
	Provisional	US 1999-166100P	19991117
	CIP of	US 1999-473510	19991228
	CIP of	US 2000-502404	20000210
	Div ex	US 2000-625399	20000726
		US 2003-622743	20030721

FILING DETAILS:

PATENT NO	KIND	PATENT NO
US 2004018546	A1 CIP of	US 6413228
	Div ex	US 6610484
	CIP of	US 6638727
	CIP of	US 6642010

PRIORITY APPLN. INFO: US 2003-622743 20030721; US
1999-117281P 19990126; US
1999-313463 19990517; US
1999-166100P 19991117; US
1999-473510 19991228; US
2000-502404 20000210; US
2000-625399 20000726

AN 2004-122028 [12] WPIDS

CR 2000-475722 [41]; 2000-491087 [43]; 2000-491116 [43]; 2000-491166 [43]; 2000-572155 [53]; 2001-016296 [02]; 2002-282788 [33]; 2002-507086 [54]; 2002-682228 [73]; 2002-723387 [78]; 2003-030038 [02]; 2003-229203 [22]

AB US2004018546 A UPAB: 20040218

NOVELTY - Identifying a patient having breast cancer or breast precancer comprising providing a ductal fluid sample from one duct of a breast of a patient, the fluid not mixed with ductal fluid from any other duct of the breast; and examining the ductal fluid sample to determine the presence of a marker, is new.

DETAILED DESCRIPTION - Identifying a patient having breast cancer or breast precancer comprising providing a ductal fluid sample from one duct of a breast of a patient, the fluid not mixed with ductal fluid from any other duct of the breast; and examining the ductal fluid sample to determine the presence of a marker, is new. The marker comprises a protein, a polypeptide, a peptide, a nucleic acid, a polynucleotide, an mRNA, a small organic molecule, a lipid, a fat, a glycoprotein, a glycopeptide, a carbohydrate, an oligosaccharide, a chromosomal abnormality, a whole cell having a marker molecule, a particle, a secreted molecule, an intracellular molecule, or a complex of a plurality of molecules.

AN INDEPENDENT CLAIM is also included for the system for diagnosing breast cancer or precancer, comprising a tool to retrieve ductal fluid from a breast duct, and instructions for use to determine the presence of the marker.

USE - The method and system are useful in diagnosing or detecting breast cancer and breast precancer in humans.

Dwg.0/0

L6 ANSWER 9 OF 12 Elsevier BIOBASE COPYRIGHT 2006 Elsevier Science B.V. on STN

ACCESSION NUMBER: 2004231088 ESBIOBASE <<LOGINID::20060930>>

TITLE: Changes in cyclic nucleotide phosphodiesterase activity and calmodulin concentration in heart muscle of cardiomyopathic hamsters

AUTHOR: Masunaga R.; Nagasaka A.; Sawai Y.; Hayakawa N.; Nakai A.; Hotta K.; Kato Y.; Hishida H.; Takahashi H.; Naka M.; Shimada Y.; Tanaka T.; Hidaka H.; Itoh M.

CORPORATE SOURCE: M. Itoh, Department of Internal Medicine, Fujita Hlth. Univ. Sch. of Medicine, Toyoake, 470 1192, Aichi, Japan.
E-mail: mituyasu@fujita-hu.ac.jp

SOURCE: Journal of Molecular and Cellular Cardiology, (2004), 37/3 (767-774), 54 reference(s)
CODEN: JMCDAY ISSN: 0022-2828

PUBLISHER ITEM IDENT.: S0022282804001828

DOCUMENT TYPE: Journal; Article

COUNTRY: United Kingdom

LANGUAGE: English

SUMMARY LANGUAGE: English

AB Cyclic nucleotides (cAMP and cGMP) ***phosphodiesterase*** (PDE) activities and expression are ***altered*** in the cardiac muscle of cardiomyopathic heart failure, and PDE inhibitors improve the abnormal muscle condition through changing the cyclic nucleotide concentration. These observations prompted us to investigate the role of calmodulin

(CaM) in the regulation of cyclic nucleotide PDE activities, and moreover to study the modulation of the PDE isozymes in heart failure, using cardiac muscles of cardiomyopathic hamster. The CaM concentrations in the heart muscle of the normal control and cardiomyopathic hamsters (each of three to four hamsters) varied with cell fraction and with the age of the animal. The CaM concentrations in the ***soluble*** fraction obtained from cardiomyopathic hamster tissue were significantly increased at 25 and 32 weeks of age (2.02 .+-. 0.62 .mu.g/mg protein (mean .+-. S.E.), and 3.21 .+-. 0.95) compared with that obtained from the control (0.60 .+-. 0.04) or cardiomyopathic (0.95 .+-. 0.12) hamsters at 8 weeks of age. The ***solubilized*** PDE isolated from the hamster heart muscle (three or four hamsters in each age) by column chromatography on diethylaminoethyl (DEAE)-cellulose revealed three peaks of activity, which may correspond to the isozymes of PDE classified recently, namely PDE I, II, and III. These three peaks of activity, particularly peak III, seen in the ***soluble*** fraction of cardiomyopathic hamster heart declined in proportion to the age of the animal compared with that of the control hamster heart. In the cGMP-PDE assay system, the concentration of CaM inhibitor W-7 required for 50% inhibition (IC.sub.5.sub.0) of PDE I, II, and III peak activities was 140, 29, and 46 .mu.M, respectively, suggesting that PDE II is more sensitive to W-7. These results suggest that ***alteration*** in these ***isozyme*** activities accompanied with changes of CaM concentration may influence the cardiac muscle contractility in cardiomyopathic hamster via changes of cyclic nucleotide concentration.

L6 ANSWER 10 OF 12 USPATFULL on STN
ACCESSION NUMBER: 2003:237767 USPATFULL <<LOGINID::20060930>>
TITLE: Genes expressed in foam cell differentiation
INVENTOR(S): Shiffman, Dov, Palo Alto, CA, UNITED STATES
Somogyi, Roland, Sydenham Ontario, CANADA
Lawn, Richard, San Francisco, CA, UNITED STATES
Seilhamer, Jeffrey J., Los Altos Hills, CA, UNITED STATES
Porter, J. Gordon, Newark, CA, UNITED STATES
Mikita, Thomas, San Francisco, CA, UNITED STATES
Tai, Julie, Cupertino, CA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2003165924	A1	20030904
APPLICATION INFO.:	US 2002-240965	A1	20021004 (10)
	WO 2001-US11128		20010404

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-60195106	20000405

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Incyte Genopmics Inc, Legal Department, 3160 Porter Drive, Palo Alto, CA, 94304
NUMBER OF CLAIMS: 19
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 2 Drawing Page(s)
LINE COUNT: 3240
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to purified polynucleotides and compositions comprising pluralities of polynucleotides that are differentially expressed during foam cell development and are associated with atherosclerosis. The present invention presents the use of the compositions as elements on a substrate, and provides methods for using the compositions and polynucleotides.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 11 OF 12 PASCAL
on STN
ACCESSION NUMBER: 1997-0014343 PASCAL <<LOGINID::20060930>>
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reserved.
TITLE (IN ENGLISH): Blunted cGMP response to agonists and enhanced glomerular cyclic 3',5'-nucleotide phosphodiesterase activities in experimental congestive heart failure
AUTHOR: SUPAPORN T.; SANDBERG S. M.; BORGESON D. D.; HEUBLEIN D. M.; LUCHNER A.; WEI C.-M.; DOUSA T. P.; BURNETT J. C. JR
CORPORATE SOURCE: Cardiorenal Research Laboratory, Mayo Clinic and Foundation, Rochester, Minnesota, United States; Renal Pathophysiology Laboratory, Mayo Clinic and Foundation, Rochester, Minnesota, United States
SOURCE: Kidney international, (1996), 50(5), 1718-1725, 55 refs.
ISSN: 0085-2538 CODEN: KDYIA5
DOCUMENT TYPE: Journal
BIBLIOGRAPHIC LEVEL: Analytic
COUNTRY: United States
LANGUAGE: English
AVAILABILITY: INIST-15906, 354000066711510330
AN 1997-0014343 PASCAL <<LOGINID::20060930>>
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AB The natriuretic peptide (NP) and nitric oxide (NO) systems are activated in congestive heart failure (CHF), resulting in increased synthesis of cGMP, which serves as a second messenger for both humoral systems. These two regulatory systems play functional roles in the preservation of glomerular filtration rate (GFR) and sodium excretion in both acute and chronic CHF. A progressive decline in glomerular responsiveness to atrial natriuretic peptide (ANP) characterizes the terminal stage of chronic CHF despite elevation of plasma ANP. ***Phosphodiesterase*** isozymes (PDEs) are integral factors in determining cellular content and accumulation of cGMP, and up-regulation of PDE activity could participate in the glomerular resistance to ANP in severe CHF. To date, characterization of possible ***alteration*** of glomerular PDE ***isozyme*** activities in CHF is unknown, as is the in vitro glomerular response to the nitric oxide- ***soluble*** guanylyl cyclase pathway. We, therefore, first determined cGMP generation in response to particulate and ***soluble*** guanylyl cyclase activation by ANP and sodium nitroprusside (SNP) in isolated glomeruli from normal (N = 6) and CHF dogs (N = 5) in which CHF was induced by rapid ventricular pacing for 18 to 28 days. Secondly, we explored the presence of major PDE isozymes in glomeruli isolated from the control and CHF dogs. When ANP or SNP (10.sup.-.sup.1.sup.0 to 10.sup.-.sup.4 M) were incubated with the suspension of isolated glomeruli, cGMP accumulation was lower by -72 to -96% with ANP and -42 to -77% with SNP in all glomerular medias obtained from CHF compared to controls. PDE hydrolyzing activity of both cAMP and cGMP were higher in the glomerular homogenates obtained from the kidneys of the CHF group (N = 5) compared to those of the control group (N = 5). We conclude that in severe chronic experimental CHF, glomerular cGMP accumulation decreases in response to both ANP and SNP, and CHF is characterized by enhanced cAMP- and cGMP-PDE activities that may participate in glomerular maladaptation to this cardiovascular syndrome.

L6 ANSWER 12 OF 12 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1996:381852 CAPLUS <<LOGINID::20060930>>
DOCUMENT NUMBER: 125:104649
TITLE: Mechanisms of satigrel (E5510), a new anti-platelet drug, in inhibiting human platelet aggregation. Selectivity and potency against prostaglandin H synthases isoenzyme activities and phosphodiesterase isoform activities
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CORPORATE SOURCE: Tsukuba Res. Labs., Eisai Co., Ltd., Ibaraki, 300-26, Japan
SOURCE: Biological & Pharmaceutical Bulletin (1996), 19(6), 828-833
CODEN: BPBLEO; ISSN: 0918-6158
PUBLISHER: Pharmaceutical Society of Japan

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Satigrel (E5510,4-cyano-5,5-bis(4-methoxyphenyl)-4-pentenoic acid) is a potent inhibitor of platelet aggregation. Like cyclooxygenase/prostaglandin H synthase (PGHS) inhibitors such as aspirin, which suppress platelet aggregation by inhibiting thromboxane A2 prodn., satigrel inhibits collagen- and arachidonic acid-induced aggregation of human platelets. In contrast to other PGHS inhibitors, satigrel, like cyclic nucleotide phosphodiesterase (PDE) inhibitors such as cilostazol, shows inhibitory activity against thrombin-induced platelet aggregation. To investigate the mechanism of the anti-platelet activity of satigrel, we examd. the selectivity and potency of satigrel against PGHS isoenzyme activities and PDE isoform activities. Two isoenzymes of PGHS are known; constitutive enzyme (PGHS1) and inducible enzyme (PGHS2). Satigrel showed inhibitory activity against PGHS1 (IC50: 0.081 .mu.M) and PGHS2 (IC50: 5.9 .mu.M), suggesting the selective inhibition of PGHS1. Indomethacin, which is a selective inhibitor of PGHS1, showed similar selectivity against PGHS isoenzymes (IC50: 0.12 .mu.M and 1.4 .mu.M, resp.). These results support that satigrel suppresses thromboxane A2 prodn. by inhibiting PGHS1. It is known that three isoenzymes of PDE exist in human platelets: type V, which specifically hydrolyzes guanosine 3;,5'-cyclic monophosphate (cGMP), Type III, which mainly hydrolyzes cAMP, and Type II, which hydrolyzes both cGMP and cAMP. We sepd., these three isoenzymes from human platelets and examd. the inhibitory activity of satigrel against each enzyme. Of the three isoenzymes, the inhibitory activity of satigrel was the most potent against Type III PDE (IC50: 15.7 .mu.M). The IC50 value for Type III corresponded with that for thrombin-induced platelet aggregation. Type V and Type II were also inhibited by satigrel (IC50: 39.8 and 62.4 .mu.M, resp.). In human platelets, satigrel increased both cAMP and cGMP levels in a dose-dependent manner (100, 300 .mu.M). In conclusion, satigrel inhibits collagen- and arachidonic acid-induced platelet aggregation through preventing thromboxane A2 synthesis by selective inhibition of the target enzyme, PGHS1, which exists in platelets. The anti-aggregating activity of satigrel against thrombin-induced aggregation may be due to elevation of the cyclic nucleotide levels through the inhibition of PDE isoenzymes.

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L1 QUE (PHOSPHODIESTERASE OR PDE4 OR PDE4D3)

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L2 175441 S L1
L3 3250 S (ISOZYME OR ISOENZYME) (S) L2
L4 177 S (MODIF? OR MUTAT? OR ALTER? OR MUTANT OR VARIANT) (S) L3
L5 12 S (AGGREGAT? OR SOLUB? OR INSOLUB?)(S)L4
L6 12 DUP REM L5 (0 DUPLICATES REMOVED)

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